

The temporal relationship between anxiety and depression in adolescence:

*A longitudinal study on symptom-development in boys
and girls.*

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Abstract

Background: Due to the common comorbidity between anxiety and depression, several attempts have been made about their temporal relationship. Research indicates that anxiety predicts depression, and depression predicts anxiety. Thus, because this relationship is unclear, the present study aimed to assess the temporal relationship between anxiety and depression symptoms from 12-13 years (t5) to 14-15 years (t6) in Norwegian adolescents. Gender differences were an additional aim throughout the study.

Method: Data were used from the Tracking Opportunities and Problems in Childhood and Adolescence (TOPP) -a longitudinal prospective population study carried out by the Norwegian Institution of Public Health. 369 (44.7% boys and 53.3% girls) adolescence completed anxiety (Coolidge Personality and Neuropsychological Inventory for Children) and depression (Short Mood and Feeling Questionnaire) measures at t5 (12-13 year of age) and t6 (14-15 years of age).

Results: As predicted, girls scored significantly higher on anxiety and depressive symptoms at both waves. Homotypic development was established from anxiety t5 to anxiety t6 and from depression t5 to depression t6. Unexpectedly, anxiety symptoms at t5 were found to be a significant independent predictor of depressive symptoms t6 in boys only. Symptoms of depression t5 were not found to be a significant predictor of anxiety symptoms t6 as predicted. This was congruent for both genders.

Conclusion: Anxiety and depressive symptoms are relatively stable constructs within a two-year time frame in early adolescence. Heterotypic predictive development was only established from anxiety to depression. As this trajectory was only found in boys, the present result suggests different developmental trajectories in boys and girls. In order to prevent clinical diagnoses of anxiety and depression from developing, it is important to identify early symptoms of anxiety and depression in youth.

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1 Introduction.

1.1. Background.

Psychological disorders have a significant impact on cognitive, emotional, and behavioural processes causing significant distress that impairs daily functioning (American Psychiatric Association, 2013). Health research carried out in all of the European countries (30) indicated an estimated number of 164.8 million who suffered from a mental disorder (Wittchen et al., 2011). In a Norwegian report, it is predicted that 1/2 of the population will develop one mental disorder during life-time, and 1/3 will meet the requirements of a diagnosable mental disorder within one year (Mykletun, Knudsen, & Mathiesen, 2009).

Anxiety and depression are the most common psychological disorders in community and primary care samples (Rouillon, 1999; Tiller, 2013; Wittchen, Beesdo, Bittner, & Goodwin, 2003), and can account for 14% (anxiety disorders) and 6.9% (depressive disorders) of psychological disorders in Europe (Olesen et al., 2012). As a consequence, anxiety and depression have been found to account for the highest proportion of indirect (sick leave, early retirement and suicide) and direct (medical supplement and treatment) costs overall in European countries (Olesen et al., 2012). In total, anxiety disorders cost European countries €74.4 billion and €113.4 billion on depressive disorders during one year (Olesen et al., 2012).

Given these estimates, preventing diagnostic disorders such as anxiety and depression from develop is important. Due to today's society, prevalence of anxiety and depression are predicted to increase (Olesen et al., 2012). One way to prevent this development from increasing is to shift attention from the clinical to the non-clinical population, and focus on anxiety and depression from a symptom level (Starr & Davila, 2012). The earlier symptom development can be identified, the better the chance of preventing a negative outcome (Jaffee et al., 2002). Thus, the current study aims to identify symptoms of anxiety and depression in a non-clinical sample. To understand development of psychological disorders, focusing on a non-clinical sample is a necessity (Cicchetti, 1984).

1.2. Emotional problems.

Based on self-report measures from longitudinal studies, prevalence of depression and anxiety symptoms have showed to have an estimation of 10-16% from 18 months to 16 years of age (Costello, Mustillo, Erkanli, Keller, & Angold, 2003), and from 15-20% in childhood

and adolescence (Helland & Mathiesen, 2009; Wichstrøm, 1999). As symptoms of anxiety and depression are not distinguished until late childhood or early adolescence, it can be difficult to differentiate between clinical and non-clinical cases of anxiety and depression in this population (Lundervold, Breivik, Posserud, Stormark, & Hysing 2013). Additionally, symptoms of anxiety and depression are common in childhood and adolescence (Costello et al., 2003; Wichstrøm, 1999), and are associated with a peak in this age-group in particular (Beesdo, Knappe, & Pine, 2009; Lundervold et al, 2013; Wichstrøm, 1999). This has contributed perceiving such symptoms in youth as a natural part of development into adulthood, and can therefore be easily overseen (Lundervold et al., 2013). Yet, 15-20% of children and adolescents with symptoms of anxiety and depression who do not meet the criteria of a clinical diagnosis still experience severe impairments in relation to their social and academic functioning (Derdikman-Eiron, et al., 2012; Lundervold et al., 2013; Wichstrøm, 1999). Thus, identifying such symptoms before they become chronic is important.

1.3. Developmental psychopathology.

Developmental psychopathology conceptually offers attention on normal and abnormal development (Cicchetti & Rogosh, 2002). Psychological disorders emerge over time, and have a developmental sequence occurring in stages (Zahn-Waxler, Klimes-Dougan, & Slattery, 2000). From a developmental psychopathology perspective it is well-demonstrated that early symptoms of anxiety and depression in childhood and adolescence can predict clinical diagnoses in adulthood (Pine, Cohen, Gurley, Brook, & Ma, 1998). Within the developmental psychopathological framework, a distinction is made between homotypic and heterotypic development. Homotypic development refers to one disorder predicting the same disorder at a subsequent time. Heterotypic development is when one disorder at one time predicts a different disorder at a subsequent time (Copeland, Shanahan, Costello, & Angold, 2009; Costello et al., 2003).

A topic of interest among researchers is the examination of the temporal relationship between anxiety and depression (Brady & Kendall, 1992; Cole, Peeke, Martin, Truglio, & Seroczynski, 1998; Starr & Davila, 2012). The question remains whether this prediction is a result of homotypic or heterotypic development over time, and whether the same pattern of developments can be applied to the clinical as well as to the non-clinical population. Another obstacle within this topic is that anxiety and depression have been found to be relatively stable

constructs over time, making predictive changes difficult to identify (Cole et al., 1998). In addition, there is considerable overlap between anxiety and depression (Cole et al., 1998; Hiller, Zaudig, & van Bose, 1989; Starr & Davila, 2012). When comparing clinical, sub-clinical and non-clinical participants, it has been found that the overlap is more extensive on a symptom level (52%) compared to a diagnostic level (29%) (Hiller et al., 1989).

1.4. Defining anxiety and depression from a dimensional approach.

Symptoms of anxiety and depression are commonly referred to as internalizing problems or emotional problems in childhood (Clark & Beck, 2010; Kovacs & Devlin, 1998; Tandon, Cardeli, & Luby, 2009). Anxiety and depression from a symptom perspective are assessed and defined following the dimensional approach (Clark & Watson, 2006). According to this approach, the focus is upon the subjective experience related to the specific psychological condition. There are no discrete disorders as represented in the categorical approach. Instead mental health problems are reflected by different set of symptoms that can be shared across different affective states (Fox, 2008).

Different symptoms are associated with specific anxiety disorders. The most common anxiety disorders in children and adolescents are separation anxiety, generalized anxiety disorder (GAD) and social anxiety (Axelson & Birmaher 2001; Costello, Egger, & Angold, 2005). Separation anxiety is associated with symptoms of worrying about being separated from attachment figures (American Psychiatry Association, 2013). GAD is characterized by uncontrollable worry that reflects symptoms like sleep disturbance, concentration difficulties, restlessness, fatigue, muscle tension and irritability (American Psychiatric Association, 2013). Symptoms associated with social anxiety are fear of social situations putting the person at risk of being negatively evaluated by others (American Psychiatric Association, 2013). Common symptoms of depressive disorders are sadness, feeling of emptiness, hopelessness, loss of interests, changes in appetite, problems sleeping, fatigue, chronically feeling of guilt and worthlessness, difficulty concentrating, and suicidal thoughts (American Psychiatric Association, 2013).

Self-reported measures are originally based on symptoms represented from taxonomies based on the categorical approach (Clark & Watson, 2006). As different psychological disorders consist of a set of symptoms, there are substantial individual differences of what symptoms are experienced, and its level of severity (Craig & Dobson, 1995; Starr & Davila, 2012). Symptoms can in addition vary within the individual from one

day to another (Starr & Davila, 2012). Thus, the dimensional approach is a valuable tool when assessing the temporal relationship between anxiety and depression from a symptom-level.

1.5. Prevalence.

1.5.1. Age of onset.

The onset of psychological disorders can take place at any time during life (Kessler et al., 1994). Research suggests that the onset of different psychological disorders varies (Kessler et al., 1994; Wittchen, Höfler, & Merikagas, 1999). The first onset of anxiety is associated with childhood, and depression with adolescence (Wittchen et al., 1999). Onset of depression in childhood is rare, but increases with age (Cooper & Goodyer, 1993; Moffitt et al., 2007; Saluja et al., 2004).

At the age of 12-13 years, Norwegian students change from primary to middle school. In addition to this social and psychological challenge, biological changes, i.e. pubertal development increase significantly from this age (Fieldman & Elliot, 1990; Spear 2000a, 2000b). Thus, symptoms of anxiety and depression are associated with a significant increase in early adolescence, and accounts for why this age group is of particular interest when examining how symptoms of anxiety and depression influence each other in development (Wichstrøm, 1999).

1.5.2. Gender differences in development.

Regarding gender differences, it is well-established that girls show a higher prevalence of symptoms of anxiety and depression compared to boys from late childhood to early adolescence (Aromstrong & Khawaja, 2002; Costello et al., 2003; Derdikman-Eiron et al., 2011). In a community sample-study, depression prevalence in adolescent girls was reported to be two to five times more frequent compared to boys (Kashani et al., 1987). Gender differences typically emerge from 12-13 years of age (Angold, Costello, & Worthman, 1998; Wichstrøm, 1999). From this age, anxiety and depressive disorders are associated with an increase in girls (Cooper & Goodyer, 1993; Derdikman-Eiron et al., 2011; Lewinsohn, Rhode, & Seeley, 1998), and are less likely to show an increase in boys (Angold & Rutter, 1992, Derdikman-Eiron et al., 2011). Another study showed gender differences when operating with clinical diagnoses of anxiety, but gender differences were not found within the non-clinical sample (Lewinsohn, Gotlib, Lewinsohn, Seeley, & Allen, 1998).

1.6. Comorbidity.

There is a considerable overlap between anxiety and depression. Based on self-reported measures, Brady and Kendall (1992) reported a correlation ranging from 0.50-0.70. Thus, it can be questioned whether symptoms of depression and anxiety first and foremost measure the same underlying construct. Within this manner, it has been argued that anxiety and depression are not independent constructs, but should be regarded as the same affective state (Brady & Kendall, 1992). Clark & Watson (2006) argued that comorbidity between anxiety and depression are too common and should first and foremost not be represented as separate entities in taxonomies such as in the Diagnostic and Statistical Manual of Mental Disorders (DSM).

Comorbidity is defined as having two concurrent disorders (Brady & Kendall, 1992; Seligman & Ollendick, 1998; Starr & Davila, 2012). Comorbidity between anxiety and depressive disorders is the most common comorbid psychological disorder (Gaynes et al., 1999), and is frequent in adolescence (Alpert, Maddocks, Rosenbaum, & Fava, 1994). From 13-18 years of age, 40 % showed a comorbid mental disorder (Merikangas et al., 1996). Although comorbidity is commonly referred to clinical disorders, it is essential to examine how anxiety and depression operates symptomatologically, as disorders first and foremost are based on a set of symptoms (Starr & Davila, 2012). Thus, assessing the temporal relationship between anxiety and depression on a symptom-level has important implications for increasing the understanding of the symptom-development as well as the comorbidity between anxiety and depressive disorders (Seligman & Ollendick, 1998; Starr & Davila, 2012). Such knowledge are crucial as comorbid psychological disorders are associated with increased personal costs due to higher symptom severity, as well as it heightens the risk of a secondary psychological disorder to develop (Bittner et al., 2004; Gaynes et al., 1999). Moreover, this has important implications for treatment of anxiety and depression (Brady & Kendall, 1992).

There are arguments declaring that comorbidity is less common when operating on a symptom-level (Cole et al., 1998). On the other hand, it is also argued that comorbidity between anxiety and depression are more common on a symptom level than on a diagnostic level, as there is a higher correlation between anxiety and depression symptomatologically (Brown, Chorpita Korotish, Barlow, 1996; Hiller et al., 1989; Lovibond & Lovibond, 1995). Thus, comorbidity is not uniquely related to clinical diagnosis, but can equally reflect anxiety symptoms of and depression (Brown et al., 1996). When examining the temporal relationship

between symptoms of anxiety and depression, there is then, a substantial need to control for comorbidity between anxiety and depression (Brady & Kendall, 1992; Copeland et al., 2009).

2. Theoretical background.

Due to the frequent comorbidity between anxiety and depression, there are several theories developed to explain their temporal relationship. Four fundamental theories on how anxiety and depression are related to each other will be presented in the following sections.

2.1. The vulnerability-stress model.

The interaction between genes and environment can make a person sensitive in developing a psychological disorder (Nugent, Tyrka, Carpenter, & Price, 2011; Rogers et al., 2013; Rutter & Silberg, 2002). The vulnerability-stress model represents two views on the temporal relationship between anxiety and depression (Wittchen et al., 2003; Wittchen, Kessler, Pfister, & Lieb, 2000). The splitter perspective views anxiety and depression as distinct psychological disorders that are independent from each other. They differ in terms of characteristic, developmental and risk factors. According to this view, anxiety predicts depression, where anxiety is considered to be a risk factor in developing a depressive disorder due to cognitive and behavioral consequences of anxiety (Wittchen et al., 2000). The lumper view represents a dimensional approach, where anxiety and depression is not perceived as two independent psychological disorders. Based on shared vulnerability risk factors and developmental features, such as genetic dispositions and characteristics, anxiety is perceived to develop prior to depression (Wittchen et al., 2000).

2.2. The tripartite model.

The Tripartite Model (Clark & Watson, 1991) is one of the most recognized models developed to explain comorbidity between anxiety and depression. The model persists that the comorbidity is caused by negative affect. Negative affect is perceived as a shared characteristic of anxiety and depression. Negative affect is referred to general negative mood and psychological distress. According to the theory, anxiety and depression can predict each other due to the negative affect. This view is consistent with the lumper perspective in where anxiety and depression are perceived as having common features. In line with the splitter perspective, anxiety and depression can be teased apart in terms of their independent unique characteristics. Uniquely, for anxiety disorders there is physiological arousal, such as nervousness, tension and shakiness. Depression is specifically associated with low positive affect that involves loss of interest and pleasure.

2.3. Biological theories.

Anxiety and depressive disorders are associated with abnormalities in neural circuits (Ressler & Mayberg, 2007) and are both characterized as stress-related disorders (Kehne & Cain, 2010). In this regard, the hypothalamic-pituitary-adrenal (HPA) system has been associated with symptoms of anxiety and depression (Kehne & Cain, 2010). The HPA-axis operates with the central nervous system (CNS), and has a significant impact upon the hormonal system. In this manner, the HPA-axis has an important impact on development of psychological disorders (Kudielka & Kirschbaum, 2005). Hypothalamic abnormality is a well-known feature of anxiety and depressive disorders. Both disorders are associated with increased levels of corticotrophin-releasing factor (CRF), a hormone secreted during a stress response (Boyer, 2000). However, there are observations demonstrating anxiety and depressive disorders shows different abnormal activity in relation to the HPA-axis.

Anxiety has been associated with hypocortisolemia which refers to anomalous low levels of cortisol in the blood stream, and depression with hypercortisolema, abnormally high levels of cortisol in the blood after dexamethasone procedure which is a synthetic glucocorticoid (Boyer, 2000). Moreover, increased glucocorticoid has been found in anxiety disorders, and decreased levels have been found in depressed patients (Boyer, 2000). Based on the observation of physiological differences in glucocorticoid levels in anxiety and depression, it can be suggested that the high levels as associated with anxiety can later predict low levels as associated with depression. In relation to understanding the temporal relationship between anxiety and depression, such observations can suggest that symptoms of anxiety predict symptoms of depression (Boyer, 2000).

2.4. Cognitive theories.

Cognitive biases are well-known features of anxiety and depressive disorders (Clark & Beck, 2010; Mathews and Mackintosh, 2000). According to Beck's (1976) Cognition Check List (CCL) theory, anxiety and depression can be discriminated by specific cognitive content. In terms of the overlap between anxiety and depression, attentional bias which has the tendency to focus on threat-related stimuli is a characteristic of anxiety disorders, but not depression (Mineka & Sutton, 1992). Depression, on the other hand, is related to memory biases, the likelihood of remembering negative events as congruent with their negative mood states. Judgmental biases have been found to be a common bias associated with both anxiety and depression (Mineka & Sutton, 1992). This suggests they have unique characteristics as well as

shared characteristics as argued by the tripartite model (Clark & Watson, 1991). One specific cognitive theory put forward to account for the temporal relationship between anxiety and depression is the helplessness-hopelessness theory. This theory persists that anxiety is associated with helplessness, and depression with hopelessness (Alloy, Kelly, Mineka, & Clements, 1990). According to the theory, helplessness predicts hopelessness, and thus anxiety is more likely to predict depression than depression to predict anxiety (Alloy et al., 1990).

Based on the vulnerability-stress model, the tripartite model, biological and cognitive theories on the temporal relationship between anxiety and depression, it points to suggest that anxiety is likely to be a precursor or a risk factor in predicting depression rather than depression to predict anxiety.

3. Previous studies.

3.1. Homotypic development.

Anxiety and depression have been found to be relatively stable constructs (Cole et al., 1998; Copeland et al., 2009; Ferdinand & Verhulst, 1995; Lewinsohn, Holm-Denoma, Small, Seeley, & Jone, 2008; Pine et al., 1998). In a three-year longitudinal study assessing children from 8-12 years, Cole et al., (1998) found a correlation of anxiety symptoms after 6 months showed 0.90 and depression 0.87. The correlation after 30 months decreased, but was still found to be strong, where anxiety showed 0.50 and depression 0.49. Relatedly, strong homotypic relationship in anxiety and depression has even been found in an eight-year longitudinal study (Ferdinand & Verhulst, 1995).

Anxiety disorders have been perceived to be more stable than depressive disorders, where pure anxiety is more common than pure depression (Mineka, Watson, & Clark, 1998). It is argued that depression is more episodic while anxiety stability may be comparable to personality traits (Prenoveau et al., 2011). However, homotypic developmental predictions have been found to differ in relation to different anxiety disorders. Stronger homotypic development has been found in panic disorder and post-traumatic stress disorder (PTSD) compared to GAD and specific phobia (Costello et al., 2003). It has been reported that heterotypic development is more frequent than homotypic development (Esbjörn, Hoeyer, Dyrborg, Leth, & Kendall, 2010). On the other hand, there are studies that have found homotypic development to be more frequent, and heterotypic development to be more common in girls compared to boys (Chaplin, Gillham & Seligman, 2009; Costello et al., 2003)

3.2. Heterotypic development.

Although homotypic development in anxiety and depressive disorders has been found, interestingly, heterotypic development is in addition established in the literature on the temporal relationship between anxiety and depression. Although, much research has been focusing on the temporal relationship between anxiety and depression, the temporality remains unclear (Kendall & Brady, 1995). Due to the DSM (American Psychiatric Association, 2013) task force, and the strong comorbidity between anxiety and depression, several attempts have been made about the heterotypic relationship between anxiety and depression (Clark & Watson, 2006). One attempt is that anxiety and depression

simultaneously co-occur and can be perceived as one disorder (Brady & Kendall, 1992; Mineka et al., 1998). The reason why they can be regarded as separate entities can be explained by symptoms experienced can vary from one day to another (Dealy, Ishiki, Avery, Wilson, & Dunner, 1981). Another attempt is to perceive them as separate entities where they developmentally influence each other (Mineka et al., 1998). Within this manner, there are studies indicating that anxiety predicts depression (Alpert et al., 1994; Bittner et al., 2004; Cole et al., 1998; Fava et al., 2000; Kourosa, Quasema, & Garbera, 2013; Starr & Dalvia, 2012; Stein et al., 2001), but also that depression predicts anxiety (Moffitt et al., 2007; Pine et al., 1998).

3.2.1. Anxiety predicts depression.

25-50 % of people with depressive disorders have been found to have an anxiety disorder, whereas 10-15 % of people with anxiety disorder have depression (Axelson & Birmaher, 2001). In addition, depressed children have been observed to indicate a high score on both anxiety and depression measures. In contrast, anxious children are less likely to establish high score on depression scales (Brady & Kendall, 1992). Thus, depression comorbid with anxiety can be regarded as more common than anxiety comorbid with depression. Such findings can propose that anxiety is more stable compared to depression (Cole et al., 1998). In support, it has been indicated that there is a higher correlation between anxiety symptoms compared to depressive symptoms (Cole et al., 1998).

From a diagnostic perspective, anxiety has been found to predict depression (Fava et al., 2000). Major depression disorder (MDD) has been found to be predicted by generalized anxiety disorder (GAD) before the age of 18 years (Alpert et al., 1994). Relatedly, Bittner et al., (2004), found specific phobia, social phobia, agoraphobia, panic disorder and GAD to be significant predictors of the first onset of MDD. As patients with MDD comorbid with anxiety disorders have been observed to be younger than MDD patients only, it supports findings indicating that anxiety disorders develops at an earlier age than MDD, and thus can serve as a risk factor in predicting MDD (Fava et al., 2000). Moreover, females are suggested to be more disposable for anxiety and depressive disorders (Breslau, Schultz, & Peterson, 1995). When gender differences in relation to MDD onset was assessed in the study of Breslau et al., (1995), it was concluded that the onset of MDD was increased in females, and could possibly be explained by previous history of anxiety.

The same pattern of findings has been established within the non-clinical population. Cole et al., (1998) tested elementary school children aged from 8-12 years in a longitudinal study. Parental and child reports of anxiety and depression were used over three years with six months assessment intervals. Parental and child reports indicated that symptoms of anxiety predicted symptoms of depression at subsequent times. This temporal relation was in addition found in a study assessing 11-14 year olds. However, anxiety symptoms to predict depressive symptoms were stronger for girls than boys (Chaplin et al., 2009). When the relationship between daily anxious and depressed mood was assessed in GAD-patients, with a past history of depression, using a 21 days diary, results showed that daily anxious mood predicted daily depressed mood. The two-day interval was found to be the strongest predictor of depressed mood (Starr & Davila, 2012). Thus, as diagnoses are made up by a set of symptoms, it may be worth emphasizing that the temporal relationship between anxiety and depression may first and foremost influence each other symptomatologically rather than diagnostically (Starr & Dalvia, 2012).

3.2.2. Depression predicts anxiety.

Depression has been found to predict anxiety when operating with clinical diagnosis of MDD and GAD. Moffitt et al., (2007) tested the temporal relationship between anxiety and depression in a 21-year longitudinal study. Subjects were assessed from 11-31 years of age. In 32 % of the cases MDD predicted GAD, whereas GAD predicted MDD in 37 % of the cases, and in the remaining percentage, GAD and MDD occurred simultaneously. It is important to note that the study did show developmental shifts in relation to the temporal relationship between GAD and MDD, respectively. There were a substantial higher percentage of GAD participants with an additional MDD, than MDD patients with GAD. However, there was an increased trend towards the opposite direction, suggesting the tendency of MDD to predict GAD increases with age. The question remains at what age this shift occur, whether this differs in terms of symptom severity, and whether this pattern of findings can be applied in terms of general symptoms of anxiety.

Consistently, Costello et al., (2003) found heterotypic development from adolescence overanxious disorder to adulthood depression, and from adolescence depression to adulthood GAD. In contrary to the findings indicating anxiety to predict depression, MDD at the age of 12 predicted GAD at the age of 18 years. The latter indicated the strongest trajectory, whereas

the prediction from overanxious disorder to depression was equally strong as depression in adolescence to predict depression in adulthood.

In the study of Cole et al., (1998) depressive symptoms were not found to later predict anxiety symptoms at a six months interval from 8-12 years of age. The GAD patients in the study of Starr and Davila (2012) did show a significant prediction of depressive symptoms to anxiety symptoms at one or two day's intervals. However, this predictive pattern was weaker than the trajectory of anxiety symptoms to predict depressive symptoms. In a six year longitudinal study, depressive symptoms was only found to predict anxiety symptoms in adolescence when mothers had a previous history of anxiety, family relationships were characterized as poor, and the child showed high levels of negative attribution (Kourosa et al., 2013). Such findings illustrate the complexity of the temporal relationship between anxiety and depression on a symptom level, and support the need for a closer examination at this relationship.

4. To conclude.

Both homotypic and heterotypic predictive development has been established within the literature on the temporal relationship between anxiety and depression. Given the mixed findings, the temporal relationship remains unclear. It is not known whether previous identified trajectories are stable throughout lifespan. In addition, most studies within the literature have been focusing on clinical diagnoses of anxiety and depression. The focus on anxiety and depression from a symptom level has increased, however research using non-clinical samples is still lacking.

5. Aims and hypotheses.

The present study was carried out to assess the temporal relationship between symptoms of anxiety and depression in a Norwegian sample from age 12-13 years (t5) to age 14-15 (t6) years. Prior to the current study, I have not found any Norwegian studies assessing the temporal relationship between anxiety and depression in this age-group in particular.

The aims of the current study are three-folded. Firstly, in order to account for the temporal relationship between symptoms of anxiety and depression, it is essential to examine the homotypic predictive development (stability) between symptoms of anxiety at t5 and t6, and depression t5 and t6. This further allows controlling for comorbidity between anxiety and depression symptoms. It is predicted that anxiety and depression symptoms will show significant homotypic development within a two year time frame. Secondly, heterotypic predictive development between anxiety t5 and depression t6, and depression t5 and anxiety t6 will then be examined. Based on the literature, it is hypothesized that symptoms of anxiety at 12-13 years of age (t5) will predict symptoms of depression at 14-15 years of age (t6). In respect of previous studies using non-clinical samples, it is hypothesized that depression symptoms at 12-13 years of age (t5) will not be a significant independent predictor of anxiety symptoms at 14-15 years of age (t6). A third aim of the current study is to assess gender differences throughout the analyses. As girls commonly report higher levels of symptoms of anxiety and depression, it is hypothesized that girls will score higher on these measures compared to boys at both waves (t5 and t6). Based on the higher prevalence of anxiety and depression in girls, the trajectory of anxiety to predict depression is hypothesized to be more likely to occur in girls compared to boys.

6. Method.

6.1. Sample and procedure.

6.1.1. The TOPP study procedure.

Data were taken from the Tracking Opportunities and Problems Study (TOPP) - a longitudinal prospective population-based study. 1081 families from 19 child health institutions in eastern Norway from 1993 were given a questionnaire when they came for the vaccination visit of their 18-month old infant. The only participation requirement was the mother's ability to read and write Norwegian. 978 of total 1081 families agreed to participate in a longitudinal study to assess the parents' and children's mental health carried out by the Norwegian Institute of Public Health. Altogether, assessments were carried out in 8 waves; when the child was 18 months (t1), 2.5 years (t2), 4.5 years (t3), 8-9 years (t4), 12-13 years (t5), 14-15 years (t6), 16-17 (t7) years and 18-19 years (t8). The parents were given a questionnaire at all waves, while the children got their own questionnaire from the age of 12-13 years (t5). All of the families participating in the study were middle class ethnic Norwegians. 28 % of the families lived in a big city, 55 % in a smaller city, and 17 % in rural area.

6.1.2. The present study.

The current study used questionnaires completed by the child from the age of 12-13 (t5), and 14-15 (t6) years of age. Participants were invited to respond to the questionnaire per post, and received a consent form prior to each assessment interval. The participants were told that the completion of the questionnaire would approximately take 30 minutes. They were further advised to be alone when answering the questionnaires due to sensitive questions, and to answer as many questions as they could. In order to follow the Regional Committee for Medical Research Ethics, all participants received an ID number. In order to protect the participant's identity, all data collected is protected by an encrypted file. Given these guidelines, only anxiety and depressive measures at t5 and t6 as well as gender and mothers demographic variables were given for the purpose of the current study. The participants were given the opportunity to withdraw from the study at any time.

6.2. Response rate.

6.2.1. T5 (12-13 years).

545 (55.7%) children of a total sample of 978 completed the items of anxiety symptoms measure by the Coolidge Personality and Neuropsychological Inventory for Children (CPNI) (Coolidge, Thede, Stewart, & Segal, 2002), and depression symptoms measured through the Short Mood and Feeling Questionnaire (SMFQ) (Angold et al., 1995). 299 (54.9%) were girls and 246 (45.1%) were boys.

6.2.2. T6 (14-15 years).

456 (46.6%) of a total of 978 adolescence completed the measures of symptoms of anxiety (CPNI) and depression (SMFQ) at t6. 254 (55.7%) were girls and 202 (44.3%) were boys.

6.2.3. Missing data.

Descriptive analyses revealed a number of missing values for each measure from the present study. Based on earlier attrition analyses of the TOPP-study data, results indicated the higher dropout rate was established within mother's educational level (Gustavson, van Soest, Karevold, & Røysamb, 2012). It was further found that the dropout did not have significant impact on the analyses (Gustavson et al., 2012). Based on the dropout from t5 to t6, missing cases were deleted in current study. Thus, the analyses carried out in the present study were based on a total of 369 respondents at t5 and t6. 165 (44.7%) were boys and 204 (55.3%) girls.

6.3. Measure.

6.3.1. Symptoms of anxiety.

11 items from the Generalized Anxiety Disorder (GAD) subscale of the CPNI (Coolidge et al., 2002) were used to measure anxiety symptoms (*see appendix I*) at both waves. From this subscale, symptoms are based on diagnostic criteria associated with symptoms of GAD, separation anxiety and social anxiety from Axis I DSM-IV (American Psychiatric Association, 1994) diagnostic criteria. Participants were asked to respond to the statements based on how they had felt during the two past months. Symptoms of anxiety t5 were assessed

on a 4-point scale ranging from 1-4 (not true-always true). Anxiety symptoms t6 were represented from 0-3 (almost never-almost always). For statistical comparisons, the scale at t6 was recoded to 1-4 (almost never-almost always). At both waves, higher scores indicate higher symptoms of anxiety.

The scale was earlier translated and back-translated in another Norwegian study (Kristensen & Torgersen, 2007). The CPNI showed a satisfactory Cronbach's Alpha Coefficient above .7 (Pallant, 2007; Tabachnick & Fidell, 2014), at t5.84 ($N=369$) and at t6 .86 ($N=363$).

6.3.2. *Symptoms of depression.*

Symptoms of depression were assessed using the (SMFQ) (Angold et al., 1995), the short version of Mood and Feeling Questionnaire (MFQ) (*see appendix II*). MFQ was developed to assess symptoms of depression from 6-17 years of age (Angold, Costello, Pickles, Winder, & Silva, 1987), and follows the DSM-IV (DSM-III-R) symptom criteria of major depressive disorder (American Psychiatric Association, 1994). The SMFQ can be used for screening as well as for clinical purposes of depression (Richter & Sund, 2013), and can thus be used in a clinical as well as in a non-clinical population (Lundervold, et al., 2013). The long version of the MFQ has been translated and back-translated from Norwegian to English by Sund, Larsson, and Wichstrøm (2001).

In the current study, 12 of 13 items from the SMFQ were presented. From the original SMFQ, 'I found it hard to think properly or concentrate' was excluded from the present version of the SMFQ because another scale already included a similar item. The subjects were told to respond to the statements based on how they had felt during the past two weeks. At t6, the depression scoring scale was recoded to fit the scoring scale at t5, and for statistical analyses was changed from 0-3 (not true-true) to 1-4 (not true-true). Hence, higher score indicated higher levels of symptoms associated with depression at both waves. Cronbach's alpha showed .86 ($N=369$) of the 12 items at t5, and .88 ($N=364$) at t6, both satisfactory $< .70$ (Pallant, 2007; Tabachnick & Fidell, 2014).

7. Results.

Data was entered into the IBM Statistical Package for Social Sciences (SPSS) 21. Based on the hypotheses, descriptive, *t*-tests, correlation and simple regression analyses were carried out. Number of items completed was used to obtain the overall mean score for each participant. To use available data, and to increase statistical power, a minimum cut-off score of 6 out of a total of 12 items was made for the SMFQ, and a minimum cut-off score of 5 out of a total of 11 items were made for the CPNI. Only participants who responded above this cut-off were used in the statistical analyses.

7.1. Preliminary analyses.

7.1.1. Normality.

It was established that skewness and kurtosis of the current data did not meet the requirements of normal distribution (> 0) (Tabachnick & Fidell, 2014). The majority of the scores were grouped to the left of the histogram, indicating a positively skewed data (Pallant, 2007; Tabachnick & Fidell, 2014). One suitable option to deal with this statistical issue is to use logarithm transformation of data (Tabachnick & Fidell, 2014). Logarithm transformation only marginally improved the distribution, and a decision was made not to use the transformed data. Also, transformation of data reduces statistical power, manipulates the natural mean and variance, and ignores outliers that are equally important as non-outliers (Grissom, 2000; Leech & Onwuegbuzie, 2002).

7.1.2. Outliers.

Potential outliers were identified in the present data. To assess how much the outliers had an impact on the analyses, the 5% trimmed mean was used. There were no major differences between the original mean and the trimmed mean. Removal or transformation of extreme scores created more outliers, and a decision was made to include all values for further analyses.

7.1.3. In summary.

As neither transformation of data, nor transformation of extreme scores was adequate, it can be argued that the scores identified in the natural data are very well representative of the Norwegian community based-sample. Thus, only the raw scores are used for the analyses throughout.

7.2. Descriptive analyses.

Based on the total means displayed in *table 1*, paired sample *t*-tests showed that anxiety symptoms at 12-13 years of age were significantly higher than symptoms of anxiety at 14-15 years of age, $t(368) = 9.26, p < .001$. There was no significant difference in depressive symptoms score from t5 to t6, $t(368) = 1.81, p = .072$.

Table 1: Total ($N = 369$) range, minimum and maximum score, mean, standard deviation, skewness and kurtosis of symptoms of anxiety (CPNI) and depression (SMFQ) at t5 (12-13 years) and t6 (14-15 years).

Variable	Range	Min	Max	Mean	Standard deviation	Skewness	Kurtosis
Anxiety t5	1- 4	1	3.82	1.55	.49	1.23	1.96
Depression t5	1- 3	1	2.83	1.31	.35	1.72	2.68
Anxiety t6	1-4	1	3.18	1.31	.38	1.72	3.11
Depression t6	1-3	1	2.92	1.35	.37	1.73	3.04

7.2.1. Between-gender differences.

Based on the means represented in *table 2*, independent sample *t*-tests revealed that girls scored significantly higher than boys on anxiety symptoms t5, $t(367) = 2.25, p = .025$, and depression symptoms at t5, $t(366.568) = 2.98, p = .003$. The same pattern of findings was found at t6. Girls scored significantly higher on anxiety symptoms, $t(366.196) = 3.22, p = .001$, and depression symptoms, $t(363) = 5.36, p < .001$ compared to boys.

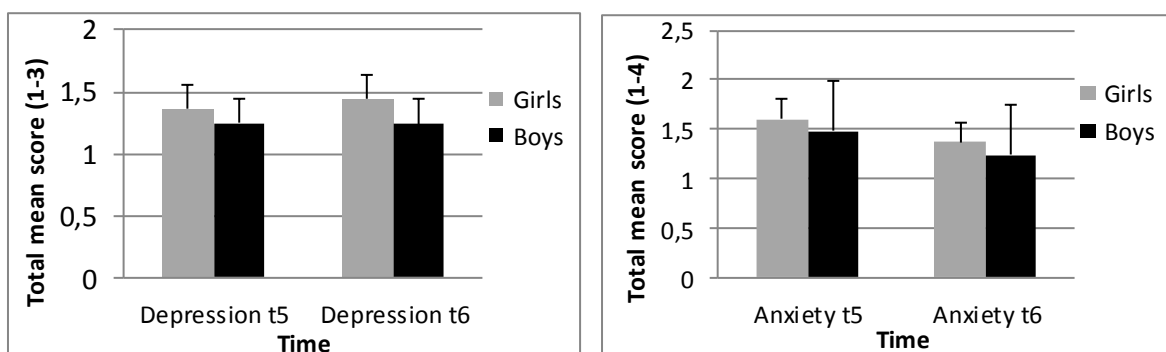
Table 2: Mean, standard deviation, minimum and maximum score, skewness and kurtosis of symptoms of anxiety and depression at t5 and t6 separated by gender. Girls: $n=204$. Boys: $n=165$.

Measures:	Anxiety t5		Depression t5		Anxiety t6		Depression t6	
Gender	<u>Girls</u>	<u>Boys</u>	<u>Girls</u>	<u>Boys</u>	<u>Girls</u>	<u>Boys</u>	<u>Girls</u>	<u>Boys</u>
Mean	1.60	1.49	1.36	1.25	1.37	1.24	1.44	1.24
Standard deviation	.48	.50	.37	.31	.40	.34	.40	.29
Min-max score	1-3.45	1-2.82	1-2.83	1-2.58	1-2.73	1-3.18	1-2.92	1-2.92
Skewness	.91	1.68	.42	2.25	1.45	2.12	1.33	2.62
Kurtosis	.53	4.15	1.55	5.45	1.60	6.89	1.45	9.02

7.2.2. Within-gender differences.

Based on the mean score for each gender on each measure displayed in *figure 1*, independent sample t -tests indicated that girls showed a significant increase in depression symptoms from t5 to t6, $t(203) = 2.62$, $p = .010$. No significant difference was found in boys, $t(164) = .42$, $p = .672$. In both genders, anxiety symptoms decreased significantly from t5 to t6, $t(203) = 6.93$, $p < .001$ (girls) and $t(164) = 6.13$, $p < .001$ (boys).

Figure 1: Graphical representation of anxiety and depression scores at t5 and t6 separated by gender. Girls: $n=204$. Boys: $n=165$.



7.3. Pearson correlation coefficient.

Table 3 shows that all variables indicated a positively significant ($p < .001$) relationship. Strongest relationship was found between depression t5 and depression t6. Depression t5 was further slightly stronger related to anxiety t5 than depression t6. Weakest relationship was revealed between anxiety t5 and depression t6, and depression t5 and anxiety t6.

Table 3: Total ($N = 369$) Person correlation coefficients between anxiety symptom-score and depression-symptom score at t5 (12-13 years) and t6 (14-15 years).

Variables:	Anxiety t5	Depression t6	Anxiety t6
Depression t5	.418***	.416***	.238***
Anxiety t5		.266***	.372***
Depression t6			.582***

*** Significant at $p < .001$ -level (two-tailed).

Separate correlational analyses were performed for each gender. A Fisher's r to z -transformation indicated non-significant differences between girls and boys in relation to the correlations between anxiety and depression measures at t5 and t6 ($p > .05$) (Preacher, 2002).

7.4. Simple linear regression analyses.

Six simple linear regression analyses were performed to examine homotypic and heterotypic developments in anxiety and depression symptoms at t5 and t6. As gender differences were identified by earlier analysis, split file was in addition used to assess these trajectories separately for boys and girls.

7.4.1. *Is anxiety t5 a significant independent predictor of depression t6 while controlling for depression t5? How stable is depression from t5 to t6?*

In the first simple regression analysis, depression t6 was entered as the dependent variable, and depression t5 and anxiety t5 as the independent variables. The overall regression was statistically significant $F(2,366) = 41.17$, $p < .001$, and overall accounted for 18.4% of the

variance in depression t6. Both anxiety t5 and depression t5 were found to be significant independent predictors of depression t6 (*table 6*).

Table 6: Total ($N=369$) standardized and unstandardized coefficients for standard linear regression with symptoms of depression t6 as the dependent variable (criterion), and depression t5 and anxiety t5 as the independent variables (predictors).

Predictors:	Standardized B	Unstandardized β
Depression t5	.39	.37***
Anxiety t5	.08	.11* ($p=.032$)

***Significant at $p < .001$ level, **Significant at $p < .001$ level, *Significant at $p < .05$ level (two-tailed).

7.4.2. Are there gender differences in relation to the temporal relationship between anxiety t5 and depression t6 while controlling for depression t5?

For girls, the overall regression was significant, $F(2.201) = 23.43$, $p < .001$, where the predictor variables could explain 18.9% of the explained variance on depression t6. For girls, only depression t5 was found to be a significant independent predictor of depression t6 (*table 7*). For boys, the overall regression was significant, $F(2.162) = 12.04$, $p < .001$ where the predictors could account for 12.9 % of the variance in depression t6. Depression t5 and anxiety t5 was found to be significant independent predictors of depression t6 (*table 7*).

Table 7: Standardized and unstandardized coefficients for standard linear regression with symptoms of depression t6 as the dependent variable (criterion), and depression t5 and anxiety t5 as the independent variables (predictors) by gender (Girls: $n= 204$. Boys: $n=165$).

	<u>Girls</u>		<u>Boys</u>	
Predictors:	Standardized B	Unstandardized β	Standardized B	Unstandardized β
Depression t5	.43	.40***	.25	.26***
Anxiety t5	.05	.06 ($p=.351$)	.09	.17 ($p=.037$)*

***Significant at $p < .001$ level, **Significant at $p < .001$ level, *Significant at $p < .05$ level (two-tailed).

7.4.3. *Is depression t5 a significant independent predictor of anxiety t6 while controlling for anxiety t5? How stable is anxiety from t5 to t6?*

In the simple regression analysis, anxiety t6 was performed as the dependent variable, and depression t5 and anxiety t5 as the independent variables. The regression analysis was significant, $F(2.366) = 31.43$, $p < .001$, and accounted for 14.7% of the explained variance on anxiety t6. Only anxiety t5 was found to be a significant independent predictor of anxiety t6 (table 8).

Table 8: Total ($N=368$) standardized and unstandardized coefficients for standard linear regression with symptoms of anxiety t6 as the dependent variable (criterion), depression t5 and anxiety t5 as the independent variables (predictors).

Predictors	Standardized B	Unstandardized β
Depression t5	.11	.10 ($p = .059$)
Anxiety t5	.25	.33***

***Significant at $p < .001$ level, **Significant at $p < .001$ level, *Significant at $p < .05$ level (two-tailed).

7.4.4. *Are there gender differences in relation to the temporal relationship between depression t5 and anxiety t6 while controlling for anxiety t5?*

For girls the regression analysis was significant, $F(2.201) = 19.47$, $p < .001$, where the predictor variables could explain 16.2% of the variance in anxiety t6. Only anxiety t5 was found to be a significant independent predictor of anxiety t6 (table 9). The overall regression analysis was significant for boys, $F(2.162) = 10.09$, $p < .001$, and the predictor variables accounted for 11.1% of the variance in anxiety t6. Anxiety t5 was only found to be a significant independent predictor of anxiety t6 (table 9).

Table 9: Standardized and unstandardized coefficients for standard linear regression with symptoms of anxiety t6 as the dependent variable (criterion), and depression t5 and anxiety t5 as the independent variables (predictors) by gender (Girls: $n=204$. Boys: $n=165$).

	<u>Girls</u>		<u>Boys</u>	
Predictors	Standardized B	Unstandardized β	Standardized B	Unstandardized β
Depression t5	.05	.04 ($p=.526$)	.15	.14 ($p=.083$)
Anxiety t5	.31	.38***	.17	.25***

***Significant at $p < .001$ level, **Significant at $p < .01$ level, *Significant at $p < .05$ level (two-tailed).

8. Discussion.

8.1. Background and aims of the present study.

Based on the comorbidity between anxiety and depression, several attempts have been developed to explain this relation. One attempt is the focus on the primary and secondary disorder, where one is believed to predict the other. Thus, the present study was conducted to examine whether anxiety symptoms at t5 (12-13 years of age) could independently predict depressive symptoms at t6 (14-15 years of age) while controlling for depressive symptoms t5, whether depression t5 could predict anxiety t6 while controlling for anxiety symptoms t5, and relatedly whether homotypic predictive relationships could be found in anxiety and depression symptoms from t5 to t6. Due to commonly gender differences in relation to internalizing problems, examining gender differences on the temporal relationship between anxiety and depression was an additional aim throughout the study.

8.2. Summary of results.

In line with the hypothesis, homotypic predictive developments were found from anxiety t5 to t6 and from depression t5 to t6, and were established in both genders. In addition, anxiety symptoms at t5 were found to be a significant independent predictor of depression symptoms t6 while controlling for depression symptoms at t5. Against the predictions, this trajectory was however only established in boys, and was not found in girls. Depression symptoms at t5 were not found to be a significant independent predictor of anxiety t6 while controlling for anxiety t5 symptoms, as hypothesized and this trajectory was established in both genders.

In relation to gender differences, as hypothesized, girls scored significantly higher on anxiety and depressive symptoms than boys at both waves. A further finding was a significant decrease in anxiety symptoms from t5 (12-13 years of age) to t6 (14-15 years of age) for both genders. Girls showed a significant increase in depression symptoms from t5 to t6. Depression symptoms did not differ significantly from t5 to t6 in boys.

In order to explain these results, the discussion will be separated by gender differences, homotypic and heterotypic development. Within the gender differences section, the differences in relation to the mean scores in boys and girls will be accounted for, in particular the decrease in anxiety symptoms from t5 to t6 in both gender, why girls scored significantly higher on anxiety and depressive scores, and why depressive symptoms

increased significantly from t5 to t6 in girls and remained stable in boys from t5 to t6. In the homotypic development section, the correlation and the regression analysis looking at how stable anxiety and depression are within a two-year time-frame will be discussed in relation to previous studies. In the heterotypic section, the gender differences in relation to the finding where anxiety was found to be a significant independent predictor in boys will be accounted for in the relation to previous studies and theories predicting this trajectory. As the present study did not find depression symptoms to be a significant independent predictor of anxiety symptoms, this will be discussed in relation to previous studies establishing this heterotypic relationship in particular.

8.3. Gender differences.

The significant decrease in anxiety symptoms from t5 to t6 in both genders is in line with previous research. Van Oort, Greaves-Lord, Verhulst, Ormel, & Huizink (2009) found anxiety symptoms to decrease from early adolescence, and then increased from middle to late adolescence. Consistently, Costello et al., (2003) found social phobia, GAD, panic disorder and separation anxiety to decrease from 9-10 years old to 12-13 years old. Moreover, Beesdo et al., (2009) indicated that anxiety decreases with age, and then may be perceived as a risk factor or a precursor in developing another disorder. Thus, this result may point to suggest a similar developmental pattern in anxiety in relation to diagnosis and symptoms in boys and girls, and this may implicate that anxiety symptoms is expressed differently throughout development as viewed by the lumpers perspective (Wittchen et al., 2003). It can then be proposed that anxiety and depression share vulnerability risks such as negative affect as viewed by the tripartite model (Clark & Watson, 1991), and biological and cognitive factors. Thus, depression may replace anxiety symptoms in early adolescence (Wittchen et al., 2000). Within this manner, this can further implicate that anxiety is a risk factor in developing depression, as biological and cognitive features associated with anxiety have been proposed to predict depression.

The significant gender difference is consistent with previous studies establishing girls to score significantly higher on depression and anxiety scores, and is less likely to increase in boys from late childhood to early adolescence (Angold et al., 1998; Angold & Rutter 1992) both symptomatologically (Aromstrong & Khawaja, 2002; Costello et al., 2003; Derdikan-Eiron et al., 2011; Kashani et al., 1987; Lewinsohn et al., 1998; Lundervold et al., 2013) and

clinically (Abbo et al., 2013; Angold & Rutter, 1992; Breslau et al., 1995; Cooper & Goodyer, 1993; Sund, Larson, & Wichstrøm, 2011).

Different assumptions and theories have been developed to explain the gender imbalance in relation to anxiety and depression prevalence. For instance, internalizing problems are more frequent in girls (Zahn-Waxler, Crick, Shirtcliff, & Wood, 2006), whereas externalizing problems is more common in boys (Zahn-Waxler, et al., 2006). Theories have argued that gender differences in relation to anxiety and depression prevalence first and foremost reflects differences operating on a genetic-level. Within this manner, an evolutionary difference may account for different developmental pathways, risk factors, and disorder-manifestation, as well as different coping mechanisms applied in boys and girls (Zahn-Waxler et al., 2006). Along these lines, Lewinsohn et al., (1998) examined psychosocial factors in relation to gender differences in anxiety. As none of the different psychosocial factors that were examined in their study were found to account for the discrepancies between boys and girls, the researchers concluded that the most valuable explanation must be due to different genetic features. It has further been suggested that this may also be a valuable explanation of gender differences in depression (Lewinsohn et al., 1994). In support, a variety of research has indicated that being a female is one risk factor in developing anxiety and depressive disorders (Lewinsohn et al., 1998; Nolen-Hoeksema 1990; Wittchen et al., 2000).

There are in addition other factors that can account for the gender differences in anxiety and depression symptoms in early adolescence. Well-being has been observed to decrease in girls and have been associated with an increase in boys from adolescence (Block & Robins, 1993), and may be linked to early puberty in girls. Gender-stereotypes may also be a valuable explanation as to why girls score significantly higher on anxiety and depressive measures (Wichstøm, 1999). Girls are perceived as more emotional compared to boys, and, as a result are treated differently by caregivers (Zahn-Waxler et al., 2006). In an Australian study assessing a non-clinical sample found that gender differences in anxiety prevalence was not explained by genetic sensitivity to anxiety disorders, but instead the cognitive attribution style in relation to the anxiety symptoms (Armstrong & Khawaja, 2002). This is congruent with studies examining cognitive attributional styles in relation to depression (Nolen-Hoeksema, Grayson, & Larson, 1999). To illustrate, in terms of cognitive theories, rumination is a common feature of depression (Nolen-Hoeksema et al., 1999). As girls have been found to ruminate more than boys this may be one factor explaining the significant increase in depression symptoms in girls and the stable levels of depression in boys (Nolen-Hoeksema et al., 1999). Another finding concluded that girls experience more interpersonal stress that is a

feature associated with depression (Shih, Eberhart, Hammen, & Brennan, 2006). As consistent with the vulnerability-stress model, development of psychological conditions is a result of the interaction between genes and environmental factors (Nugent et al., 2011; Rogers et al., 2013; Rutter & Silberg, 2002). This implies that not all girls will experience anxiety and depressive symptoms, rather, genetically disposable girls and negative life events make girls more susceptible of anxiety and depression symptoms (Silberg, Rutter, Neale, & Neaves, 2001).

Moreover, it has been argued that response bias may account for the well-established gender differences in anxiety and depression symptoms when self-reported measures are used (Sigmon et al., 2005). In this regard, girls are more likely to report depression and anxiety compared to boys (Armstrong & Khawaja, 2002; Derdikman-Eiron et al., 2011). It is important to note that this may not necessarily mean girls are more depressed or anxious than boys (Saljua et al., 2004), rather it can suggest they may be more conscious about their emotions.

In conclusion, there is not one single factor that can serve to explain gender differences in anxiety and depression symptom prevalence, but there is an interaction between a number of variables connecting to genetic and environmental factors. It is worth noting that the present study indicates that gender difference operates on a symptom-level in relation to prevalence of anxiety and depression. And, furthermore this development may be differently established in boys and girls.

8.4. Homotypic development.

8.4.1. How stable is anxiety and depression symptoms from t5 to t6?

Both anxiety and depression symptoms was found to be stable constructs from 12-13 years of age to 14-15 years of age in both genders. This is consistent with previous studies establishing anxiety and depression symptoms to be stable constructs over time (Cole et al., 1998; Costello et al., 2003; Ferdinand & Verhulst, 1995; Lewinsohn, Holm-Denoma, Small, Seeley, & Joiner, 2008). Specifically, the current study is congruent with the study of Cole et al., (1998), where it was found that the correlation of anxiety symptoms after 6 months was 0.90 and 0.59 after 30 months based on child reports. The correlation in depressive symptoms after 6 months was found to be 0.87 and 0.49 after 30 months. The correlations found in the present study was weaker than the relations identified in the study of Cole et al., (1998), but still after

a two-year period they were found to be moderate-strong, and can be interpreted as being stable constructs over time in early adolescence.

The current study established depression symptoms from t5 to t6 to be more stable than anxiety symptoms from t5 to t6, and is inconsistent with previous studies. In the study of Cole et al., (1998), anxiety was found to be slightly more stable than depression after 6 and 30 months. The sample used in the study of Cole et al., (1998) was aged from 8-12 years, whereas the age-group in the present study ranged from 12-13 and 14-15 years of age, and may be a possible explanation in relation to this inconsistency. In support of this argument, depression is commonly associated to increase with age (Cooper & Goodyer, 1993; Moffitt et al., 2007), and is in particular linked with an increase in early adolescence (Wittchen et al., 1999). Thus, it may point to suggest that depressive symptoms are more stable than anxiety symptoms in early adolescence compared to childhood. Previous studies that indicate anxiety to be more stable than depression over time have been assessing clinical samples. For instance, in terms of comorbidity between anxiety and depression, it has been found that pure anxiety is more common than pure depression (Axelson & Birmaher, 2001). This pattern of finding has been used to explain why depression comorbid with anxiety is more common than anxiety comorbid with depression (Axelson & Birmaher, 2001; Brady & Kendall, 1992). In relation to the non-clinical sample used in the current study, it may be proposed that depression is more stable than anxiety when operating with symptoms rather than on diagnosis in early adolescence.

The regression analyses in the current study established that homotypic predictive development was stronger than the heterotypic predictive development from t5 to t6, and this was found in boys and girls. This was also concluded in the study of Costello et al., (2003), where adolescents from 9-13 years were assessed until the age of 16 years. Homotypic development was in addition established in a non-clinical study assessing symptoms of anxiety and depression in 11-14 year olds (Chaplin et al., 2009). Thus, due to little change in anxiety and depression over time, heterotypic predictive relationships in early adolescence can be difficult to identify (Cole et al., 1998).

8.5. Heterotypic development.

8.5.1. *Is anxiety symptoms t5 a significant independent predictor of depressive symptoms t6 while controlling for depression t5?*

Despite the stable constructs in anxiety and depression symptoms over a two-year time period, heterotypic prediction was also found in the present study. Anxiety symptoms at 12-13 (t5) years of age were found to be a significant independent predictor of depression symptoms at 14-15 years of age (t6) even when controlling for symptoms of depression t5. This pattern of finding is consistent with clinical (Alpert et al., 1994; Bittner et al., 2004; Brady & Kendall, 1992; Breslau et al., 1995; Fava et al., 2000; Starr & Davila, 2012) and non-clinical studies (Cole et al., 1998; Chaplin et al., 2009). Moreover, this trajectory is consistent with, and extends previous studies indicating anxiety to predict depression with six months (Cole et al., 1998) and two days assessment intervals (Starr & Davila, 2012). Inconsistent with the hypothesis, this developmental pathway was only established in boys, and was not found in girls. A previous study found heterogenetic predictive development in relation to different psychological disorders, including anxiety and depression to be more common in girls than boys (Costello et al., 2003). When gender differences were directly examined in relation to the trajectory of anxiety symptoms to predict depression symptoms, this heterogenetic development was found to be stronger in girls than boys (Chaplin et al., 2009).

The reason why anxiety was found to predict depression in boys can be explained in terms of anxiety symptoms have been found to be associated with more negative consequences in boys compared to girls. Derdikman-Eiron et al., (2012) concluded when well-being, self-esteem and psychosocial functioning was examined in relation to symptoms of anxiety and depression it was found that anxiety had a stronger negative impact in these areas in boys compared to girls. As a consequence, boys may feel more distress despite of indicating lower scores on anxiety symptoms compared to girls. In support of this assumption, it has been found that boys experience more chronic stress and girls more episodic stress (Shih et al., 2006). It is well-established that stress has a significant impact on anxiety and depression (Khene & Cain, 2010), where anxiety, depression and stress scores are positively significant related (Al-Gelban, Al-Amri, & Mostafa, 2009). Based on the results of the current study, it can be suggested that the degree of stress associated with anxiety symptoms influences whether anxiety is a significant predictor of depression symptoms (Wittchen et al., 2000).

This developmental trajectory where anxiety predicted depression, it can indicate that anxiety and depression are distinct constructs where anxiety put development of depression at risk as argued by the splitter perspective (Wittchen et al., 2000). This can be explained in terms of biological and cognitive theories. From biological observations, the high glucocorticoid levels in anxiety can over time manifest in low levels of glucocorticoid as

associated with depression (Boyer, 2000). In relation to the findings of the HPA-axis activity, the more distress experienced due to anxiety symptoms may over time reach physiological activation associated with depression (Boyer, 2000; Zahn-Waxler et al., 2006). Thus, it suggests that anxiety symptoms must influence the physiological processes to a significant extent in order to predict depression symptoms within a two-year time frame. In order to account for why anxiety was found to be a significant independent predictor in depression to in boys only, there are studies implicating that there are gender differences in relation to stress-responses. It has been observed that males showed a stronger HPA-axis activation to a stress-induced task compared to females (Kudielka, Buske-Kirschbaum, Hellhammer, & Kirschbaum, 2004)

In terms of cognitive theories, anxiety is regarded as a risk factor due to cognitive content. As introduced by Alloy et al., (1990), anxiety is likely to predict depression because hopelessness that is associated with depression is less likely to occur without helplessness as associated with anxiety. In support, it has been found that acute stress has been associated with more fear and helplessness in males compared to females (Heinsbroek, Van Haaren, van de Poll, & Steenbergen, 1991; Steenberg, Heinsbroek, Van Harren, vande Poll, 1990), and may indicate that stress among boys put them at risk of feeling helpless leading anxiety to predict depression.

In relation to the tripartite model (Clark & Watson, 1991), this temporal pattern can alternatively be explained in terms of the negative affect that is believed to bridge the temporal relationship between anxiety and depression. As a result, negative affect may be stronger when anxiety symptoms are experienced as more stressful. From this perspective, it can be interpreted that negative affect displays differently throughout development, where anxiety develops prior to depression as suggested by the lumpers perspective (Wittchen et al., 2000). In relation to the lumpers perspective, it can be suggested that negative affect is expressed as anxiety in childhood, and depression in adulthood, allowing anxiety to predict depression in early adolescence. This may further indicate that even symptomatologically, anxiety and depression's negative affect is met to a significant leading this trajectory to develop.

The results of the present study are consistent with the possibility that genetic differences in boys and girls can establish different developmental trajectories in relation to psychological disorders, and different risk factors (Zahn-Waxler et al., 2006). In line with this assumption, Duggal, Carlson, Sroufe, & Egeland (2001) found that childhood and adolescence depression may be explained by different risk factors in boys and girls. Mother's

depression was stronger related to depression in adolescent girls compared to boys. For boys, early care was found to be the strongest predictor of depression onset in adolescent. And, another support, Castealo & Kröner-Herwig (2013) established different developmental trajectories in boys and girls regarding development of depression symptoms. Relatedly, this was also a conclusion in the study of Chaplin et al., (2009) where worry and oversensitivity in relation to anxiety symptoms were stronger for girls than boys. Thus, the reason why anxiety t5 was not found to be a significant independent predictor of depression t6 in girls may be caused by different developmental pathways regarding the temporal relationship between anxiety and depression symptoms in boys and girls. As a result, there may be other risk factors than anxiety symptoms that better predicts depressive symptoms in non-clinical girls from 12-13 to 14-15 years of age. Along this line, it has been argued that girls' earlier onset of anxiety and increase in depression can be explained by early puberty (Stice, Presnell, & Bearman, 2001), where this time-period is associated with significant challenges in early adolescence compared to boys (Petersen, Sarigiani, & Kennedy, 1991).

8.5.2. Is depression symptoms t5 a significant independent predictor of anxiety symptoms t6 while controlling for anxiety t5?

Results of the present study are consistent with previous research in relation to whether symptoms of depression can account to be a significant independent predictor of symptoms of anxiety at subsequent time (Cole et al., 1998). Although nearly significant, depression was still not found to be an independent predictor of anxiety t6 symptoms at one-year interval. The same developmental trajectory was found in boys and girls.

In the study of Cole et al, (1998) the opposite trajectory was established based on parental reports. Children perceived as more depressed at one time were being perceived with a decrease in anxiety symptoms at a subsequent time. Kourosa et al., (2013) only found depressive symptoms to predict anxiety symptoms when mothers had previous history of anxiety, poor family relationships and the child showed high levels of negative attributions. This can indicate that risk factors may mediate the trajectories where depression symptoms predict anxiety symptoms.

Previous studies showing that depression predict anxiety has been found in studies assessing diagnostic criteria of anxiety and depression. Hence, it can suggest that depression is more likely to predict anxiety when depression symptoms are severe. In line with this assumption, it has been found that severity is the strongest predictor of depression onset

(Bittner et al., 2004; Wittchen et al., 2000). When depression has been found to predict anxiety, this trajectory has been found with older samples (Dilsaver et al., 1992; Moffitt et al., 2007) and may only be limited to Generalized Anxiety Disorder (GAD) (Moffitt et al., 2007) and possibly social anxiety (Dilsaver, Qumar, & Del Medico, 1992). When assessing symptoms of GAD and MDD in GAD patients with past history of MDD, Starr & Davila (2012) did find a tendency of depressive symptoms to predict anxiety. It is suggested that GAD should be classified as a subtype of depressive disorders (Moffitt et al., 2007). Thus, the reason why they can influence each other can be explained by considerable symptom overlap (Starr & Davila, 2012). Massion, Warshaw, & Keller (1993) argue that GAD and MDD equally precede each other, they should be perceived as sharing the same underlying symptom. In terms of depression to predict anxiety, the same trajectory has been found with social anxiety. Dilsaver et al., (1992) found major depression to precede social anxiety. As the study of Dilsaver et al., (1992) assessed adult participants from 18 – 50 years of age, with a mean age of 36.5 years, it might be possible that in early age, anxiety precedes depression, and in older age, depression can precede anxiety, and further, that it may be limited to the clinical population in particular.

And, furthermore, this can implicate a shared negative affect as argued by the tripartite model (Clark & Watson, 1991), as well as cognitive biases, and biological features that influence each other interchangeably.

To summarize, the results of the present study indicate girls show higher anxiety and depression scores compared to boys from 12-13 to 14-15 years of age. As anxiety was only found to be a significant independent predictor of depression in boys, in line with previous research it is suggested that stress and coping-mechanisms in relation to anxiety symptoms may account for this gender difference. Thus, this can propose that different developmental pathways may be established differently in boys and girls in relation to the temporal relationship between anxiety and depression operating on a symptom-level. As depression symptoms were not found to be a significant independent predictor of anxiety symptoms, it is argued that this may only be applied to specific anxiety disorders and may only be limited to clinical diagnoses of anxiety and depression. In order to increase the understanding of the results of the present study, it is important to identify important limitations and suggest future research. This will be presented in the following sections.

9. Limitations and future research.

9.1. Methodological issues.

9.1.1. Prospective longitudinal studies.

The sample from the TOPP-study data was collected from the eastern part of Norway only, thus the sample is not representative to the general Norwegian population aged from 12-13 to 14-15 years. Furthermore, as the present study only used a non-clinical sample, the results cannot be generalized to the clinical population. Thus, in order to achieve a satisfactory picture of the temporal relationship between anxiety and depression over time, a necessity is to carry out longer prospective longitudinal studies comparing clinical and non-clinical samples (Moffitt et al., 2007).

Another problem with prospective longitudinal studies is that they identify developmental trajectories within a specific time frame (Alonso, Chatterji, He, & Kessler, 2013), and cannot serve to explain lifetime developmental trajectories. As only two assessment intervals were administered in the current study, it is not known how anxiety and depression symptoms changes with a shorter or longer assessment intervals. This can be important as anxiety and depression have found to be stable construct over time. The study of Copeland et al., (2009) indicated that childhood depression only predicted adulthood depression when anxiety and depression was comorbid in adolescence. Thus, it may suggest that anxiety and depression first and foremost represents the same underlying construct that changes through development as argued by the lumpers perspective. Based on the current study, it is not known whether symptoms of anxiety that predicts depression will later meet diagnostic criteria of anxiety or depression, or comorbidity. Whether homotypic stability from t5 to t6 will predict heterotypic development in time cannot be concluded based on the two-year assessment interval in the current study. Thus, it remains for future research to examine how anxiety and depression symptoms in early adolescence changes within shorter and larger assessment intervals.

9.1.2. Specific anxiety disorders.

Different types of anxieties were not considered separately in the current study. As the GAD subscale of the CPNI (Coolidge et al, 2002) does include symptoms associated with social anxiety, separation anxiety and generalized anxiety, the present study did not account for how

these separately can predict depression symptoms. Thus, it will be important for future research to further carry out longitudinal studies using different separate questionnaires that measure specific anxiety symptoms. Specifically, as depression and GAD has been found to influence each other diagnostically, it remains for future studies to assess whether this may in addition be applied when focusing on symptom of anxiety and depression in a non-clinical sample.

9.1.3. Self-report measure.

Self-report measures cannot always be reliable as it leads to question the reliability and validity of the measure because of different response-biases with regards to lying, exaggerating, and hiding negative emotions, wanting to represent themselves in a positive light, mood of the day and response style (Craig & Dobson, 1995; Robins & Tanck, 1987). To solve this problem, future research should be focusing on interviews (Craig & Dobson, 1995). Alternatively, in order to improve the validity and reliability, self-report measures should be administered to the youth in addition to teachers and parents (Cole et al., 1998).

9.1.4. Item overlap between anxiety and depression questionnaires.

Although, anxiety and depression share many common symptoms (Cole et al., 1998; Starr & Davila, 2012), the present study did not assess or removed overlapping items. Thus, the reason why research has found a relationship between depression and anxiety can be explained in terms of the questions asked mainly measure the same underlying structure (Clark & Watson, 1991). Then, the overlap between anxiety and depression measures may occur when the anxiety measure consists of different specific anxiety symptoms, as in the CPNI. This can specifically occur when anxiety symptoms associated with GAD are present. The high correlation between GAD and depression is a well-known phenomenon in particular (Kendall & Brady, 1992; Moffitt et al., 2007). Thus, it is important to take overlapping items into consideration when measuring how anxiety and depression influence each other.

9.1.5. Mediating variables.

The current study did not examine potential factors that can potentially mediate the temporal relationship between anxiety and depressive symptoms. As a possible explanation in relation to the different developmental trajectory in boys and girls in the current study can be accounted for biological and cognitive features, it will be of importance for future research to examine mediating variables. This will further develop an understanding in how boys and

girls differ in relation to the temporal relationship between symptoms of anxiety and depression in adolescence. Thus, the temporal relationship between anxiety and depressive symptoms in early adolescence is complex. Lastly, the present study used regression analyses on questionnaire data to examine the temporal relationship between anxiety and depression, it does not allow making any conclusions about the causal relationship between anxiety and depression (Tabachnick & Fidell, 2014; Wittchen, Beesdo et al., 2003).

9.2. Statistical issues.

Data of the present study was positively skewed, indicating the majority of the sample had generally low scores on both anxiety and depressive symptoms at both waves. This has been established with other studies using community-based samples (Angold, Erkanli, Silberg, Eaves, & Costello, 2002; Cannon & Weems, 2005). This problem is common when dealing with continuous measures such as anxiety and depression (Angold et al., 2002). However, parametric tests such as regression analyses ideally require data to be normally distributed, thus, the interpretation of the current results should then be done with care (Angold et al., 2002; Tabachnick & Fidell, 2014).

10. Conclusion and clinical implications.

Homotypic predictive development was established in the present study, where anxiety and depression symptoms were found to be relatively stable constructs within a two-year time frame in boys and girls. In addition this accounted for the strongest predictive relationship compared to heterotypic development. Despite the stable constructs of anxiety and depressive symptoms, heterotypic predictive development was established from anxiety symptoms at t5 to depression symptoms at t6. However, depression symptoms t5 were not found to be a significant independent predictor of anxiety t6. As anxiety symptoms were found to predict depressive symptoms in boys only, the current study suggests different developmental pathways in relation to anxiety and depression may be present in boys and girls. It will be important for future research to account for how girls and boys differ in relation to the temporal relationship between anxiety and depression symptoms as this has been lacking in the literature.

The current study further indicates that it is important to take early symptoms of depression and anxiety seriously. Early symptoms of anxiety may prevent development of clinical diagnoses of depression and anxiety, and comorbidity between anxiety and depression. Thus, early anxiety symptoms can be regarded as a risk or a precursor of subsequent anxiety and depression symptoms due to genetic vulnerability, biological and cognitive factors. Although girls are more likely to experience anxiety and depression symptoms, it is further important to give attention to boys' anxiety and depressive symptoms. Results of the current study should be presented to teachers, parents and other relevant people surrounded with young adolescents. This will improve the chances of identifying early symptoms of anxiety and depression, and prevent clinical diagnoses (and even other psychological disorders) in the population from developing.

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APPENDIX I

Coolidge Personality and Neuropsychological Inventory for Children (CPNI).

Barn og ungdommer kan være engstelige i perioder. Tenk på hvordan du har hatt det de siste månedene, og kryss av slik at det passer for deg:

	1 Stemmer absolutt ikke	2 Stemmer sjelden	3 Stemmer ofte	4 Stemmer alltid
Jeg blir veldig urolig når jeg må gå fra foreldrene mine eller dra hjemmefra	<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>
Jeg unngår sosiale aktiviteter fordi jeg er redd for å bli kritisert eller avvist	<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>
Jeg bekymrer meg mye for at det skal hende noe fælt med foreldrene mine	<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>
Jeg er redd for å bli forlatt og måtte passe på meg selv	<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>
Jeg bekymrer meg mye for å komme bort fra foreldrene mine eller å bli kidnappet	<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>
Jeg har forferdelige mareritt	<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>
Jeg er for mye bekymret	<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>
Jeg bekymrer meg for mye for å bli avvist eller kritisert	<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>
Jeg er redd for å gå fra foreldrene mine (som når jeg skal på skolen)	<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>
Jeg er engstelig i sosiale situasjoner fordi jeg er redd for andre mennesker	<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>
Jeg er redd for å gjøre nye ting i frykt for å dumme meg ut	<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>
Jeg henger meg så mye opp i detaljer eller f.eks. tidsplaner at jeg glemmer hva det er jeg egentlig skal gjøre	<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>
Jeg må stadig sjekke at jeg har gjort ting på den riktige måten (som at døren er låst, gymtøyet er med)	<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>
Jeg har vanskelig for å få dumme eller rare tanker ut av hodet	<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>
Jeg må tenke på spesielle måter (som på bestemte tall eller ord) for å forhindre at farlige ting skal skje	<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>
Jeg må gjøre ting om og om igjen (som å vaske hendene eller lage spesielle system i ting)	<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>
Jeg må gjøre ting på helt spesielle måter for å forhindre at farlige ting skal skje	<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>

APPENDIX II

Short Mood and Feeling Questionnaire (SMFQ).

Her er en liste over forskjellige plagsomme følelser og tanker man av og til kan ha. Tenk på de to siste ukene og kryss av for hvor ofte du har følt eller tenkt noe av det som står nedenfor: (*sett ett kryss på hver linje.*)

	1	2	3
	Stemmer ofte	Stemmer noen ganger	Stemmer ikke
Jeg var lei meg eller ulykkelig	<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>
Jeg følte meg så trøtt at jeg bare ble sittende uten å gjøre noen ting	<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>
Jeg var veldig rastløs	<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>
Jeg var ikke glad for noe	<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>
Jeg følte meg lite verdt	<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>
Jeg gråt mye	<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>
Jeg hatet meg selv	<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>
Jeg tenkte at jeg aldri kunne bli så god som andre barn	<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>
Jeg følte meg ensom	<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>
Jeg tenkte at ingen egentlig var glad i meg	<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>
Jeg følte meg som et dårlig menneske	<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>
Jeg syntes jeg gjorde alt galt	<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>